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Association Between Adult Attention Deficit/Hyperactivity Disorder and Obesity in the US Population

S. L. Pagoto¹, C. Curtin², S. C. Lemon¹, L. G. Bandini^{2,3}, K. L. Schneider¹, J. S. Bodenlos¹, and Y. Ma¹

¹University of Massachusetts Medical School, Department of Medicine, Division of Preventive and Behavioral Medicine Worcester, MA

²University of Massachusetts Medical School, Eunice Kennedy Shriver Center, Waltham, MA

³Boston University, Department of Health Sciences, Boston, MA

Abstract

Attention deficit hyperactivity disorder (ADHD) is a neurobehavioral disorder that affects approximately 2.9%–4.7% of US adults. Studies have revealed high rates of ADHD (26 – 61%) in patients seeking weight loss treatment suggesting an association between ADHD and obesity. The objective of the present study was to test the association between ADHD and overweight and obesity in the US population. Cross-sectional data from the Collaborative Psychiatric Epidemiology Surveys were used. Participants were 6,735 U.S. residents (63.9% Caucasian; 51.6% female) aged 18 to 44. A retrospective assessment of childhood ADHD and a self-report assessment of adult ADHD were administered. Diagnosis was defined by three categories: never met diagnostic criteria, met full childhood criteria with no current symptoms, and met full childhood criteria with current symptoms. The prevalence of overweight and obesity was 33.9% and 29.4%, respectively, among adults with ADHD, and 28.8% and 21.6%, respectively, among persons with no history of ADHD. Adult ADHD was associated with greater likelihood of overweight, [odds ratio (OR)=1.58; 95% confidence interval (CI)=1.05, 2.38] and obesity (OR=1.81; 95% CI=1.14, 2.64). Results were similar when adjusting for demographic characteristics and depression. Mediation analyses suggest that binge eating disorder, but not depression, partially mediates the associations between ADHD and both overweight and obesity. Results suggest that adult ADHD is associated with overweight and obesity.

Keywords

ADHD; Epidemiology; Obesity

INTRODUCTION

Once assumed to be a disorder of childhood, attention deficit hyperactivity disorder (ADHD) has been shown to persist into adulthood and affects over 9 million Americans (1). As in children, core features of adult ADHD include inattention, impulsivity, distractibility, and/or hyperactivity (2). Adults with ADHD commonly report procrastination, boredom

Corresponding author: Sherry L. Pagoto, University of Massachusetts Medical School, Department of Medicine, Division of Preventive and Behavioral Medicine, 55 Lake Avenue North, Worcester, MA 01655, (508) 856-2092 telephone, (508) 856-3840 fax, sherry.pagoto@umassmed.edu.

DISCLOSURE

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intolerance, frustration intolerance, disorganization, affect dysregulation, and heightened emotional reactivity (3). Adults with ADHD also have elevated rates of arrest histories, poor academic performance, depression, anxiety, traffic accidents, occupational problems, tobacco use, and substance abuse (4). Less research has explored health problems associated with ADHD.

Obesity affects 31% of the U.S. population (5) and certain mental disorders predict greater risk for obesity-related disease. Mental disorders characterized by appetite and/or affect dysregulation such as binge eating disorder (6) and depression (7) have been the primary targets of study. However, a few studies have revealed rates of ADHD in weight treatment-seeking adults 5–15 times higher than rates in population samples (8–11). Whether ADHD is linked to increased risk for obesity in the general adult population has not yet been explored.

ADHD may be linked to obesity in children. One study found 19.5% of 5–14 year old boys diagnosed with ADHD had body mass indexes (BMI) \geq 90th percentile and 7.2% had BMIs \geq 97th percentile (11), nearly twice as high as the general population in that age range. Consistent findings were reported in a study of children aged 8–17 who were hospitalized for obesity. Over half (57.7%) met DSM-IV criteria for ADHD (8). A study of children aged 3–18 with ADHD, 29% were at risk for overweight (BMI $>$ 85th percentile) and 17.3% for obesity (BMI $>$ 95th percentile) (12), which is two and three times higher than the general population, respectively. Finally, a population-based study revealed that among 1,429 Chinese students aged 13–17, those with ADHD symptomatology were 1.4 times as likely to be obese than lean (13).

Two studies explored the relationship between ADHD and obesity in adults. One study of obese patients seeking weight loss treatment revealed 27.4% met diagnostic criteria for ADHD, a rate six times greater than what has been reported in the general population (9). Patients with ADHD also had significantly higher BMIs and lost significantly less weight during weight loss treatment than those without ADHD. Similar findings were reported in a study of 75 severely obese women referred for weight loss treatment. About 27% reported symptoms of ADHD in both childhood and adulthood (10). Findings from these clinic-based studies suggest an association between adult ADHD and obesity. Population studies are needed because rates of psychiatric disorders are typically inflated in clinic samples. Also, the extent to which depression and binge eating disorder (BED) account for the relationship between ADHD and obesity should be explored given that each is independently associated with obesity (14, 15) and ADHD (1, 16). The purpose of the present study was to examine the association between ADHD and risk for overweight and obesity, and the degree to which the association is accounted for by depression and BED among adults sampled from the general U.S. population.

METHODS

The Collaborative Psychiatric Epidemiology Studies (CPES), sponsored by the National Institute of Mental Health, includes three nationally representative surveys of U.S. adults. These surveys were designed to provide epidemiologic data regarding mental disorders using standardized diagnostic criteria. The three surveys include the National Comorbidity Survey Replication (NCS-R), the National Study of American Life (NSAL) and the National Latino and Asian American Study of Mental Health (NLAAS) (17). Although independent surveys, the CPES were designed to allow combining of the three datasets by having shared scientific objectives, measurement tools, implementation procedures, and sampling methodologies (18). The core CPES questionnaire was based on the World Health Organization's expanded version of the Composite International Diagnostic Interview

(CIDI). The present study included only the NCS-R and the NSAL because the NLAAS did not assess ADHD.

The NCS-R was a cross-sectional survey designed to provide representative data of the non-institutionalized population of English-speaking U.S. adults age 18 and over between 2001 and 2003 (19). The NCS-R used a four-stage probability sampling method, first sampling Metropolitan Statistical Areas and counties, then area segments, followed by housing units within the area segments, and finally eligible respondents within the housing units (18, 19). The recruitment method included a mailed introductory letter and study brochure followed by a household visit by a trained interviewer to identify eligible respondents and provide informed consent. The survey consisted of face-to-face computer-assisted personal interviews (CAPI). The interview included two parts. Part 1 included the core diagnostic assessment of all 9,282 persons participating in the study (response rate 70.9%). Part 2 was administered to a subset of 5,692 respondents, including all who met lifetime diagnosis of a disorder and a probability sample of all other respondents. Part 2 included questions related to disorders of secondary interest. Diagnostic questions for ADHD were included in Part 2, and were asked only of persons age 18 to 44 ($n=3,197$) (1).

The NSAL was a cross-sectional survey conducted between 2001 and 2003 that targeted English-speaking U.S. adults who self-identified as African American ($n=3570$), of Caribbean descent ($n=1,621$) and non-Hispanic Whites living in predominantly black neighborhoods ($n=891$). A sampling method similar to that of the NCS-R was used (18, 20). Interviewers made household visits to obtain consent and complete the CAPI. The majority of CAPIs (86%) were completed face-to-face and the remaining by phone. The response rate was 71.5%. The entire sample ($n=6,082$) completed all components of the CAPI. Analyses were restricted to respondents aged 18 to 44 ($n=3,538$) to be consistent with the NCS-R. Analyses were performed using SAS version 9.1.2 and Stata version 10.

The analytic sample for this study included 6,735 respondents aged 18 to 44 years old, 3,197 from the NCS-R and 3,538 from the NSAL. In the NCS-R, only a randomly selected subsample completed questions related to eating disorders, of whom 1,672 were 18 to 44 years old. Thus, analyses including the binge eating disorder (BED) variable were conducted only among 5,210 respondents, 1,672 from the NCS-R and 3,538 from the NSAL. Because data were missing at random, imputation methods for missing data were not performed. This study was approved by the University of Massachusetts Medical School Institutional Review Board.

Measures

Respondents self-reported their height and weight as part of the core interview. BMI was calculated using the standard formula (weight in kilograms/height in meters squared). BMI was included in the CPES as a recoded six category variable: underweight ($BMI \leq 18.4$); normal weight ($BMI 18.5-24.9$), overweight ($BMI 25.0-29.9$), class I obesity ($BMI 30.0-34.9$), class II obesity ($BMI 35.0-39.9$) and class III obesity ($BMI \geq 40.0$). For the purposes of the present study, we recoded BMI into a three category variable: normal/underweight, overweight, and obese.

As described elsewhere (1), childhood ADHD was assessed using a retrospective version of the Diagnostic Interview Schedule for DSM-IV (21). Respondents who reported 6 or more symptoms of inattention or hyperactivity were administered follow-up questions regarding the remaining DSM-IV criteria. If a respondent met criteria for childhood ADHD, they were then asked whether problems persisted to the present. Clinical reappraisal interviews were performed by clinical interviewers on a subsample of participants using the Adult ADHD Clinical Diagnostic Scale (22, 23), the ADHD Rating Scale (24), and an adaptation of the

ADHD Rating Scale. Respondents were diagnosed with adult ADHD if they endorsed six or more symptoms of either inattention or hyperactivity within the last six months (DSM-IV Criterion A), two or more Criterion A symptoms before age seven (Criterion B), impairment in two or more areas of living within the past six months (Criterion C), and clinically significant impairment in at least one of these areas (Criterion D). No attempt was made to operationalize DSM-IV diagnostic hierarchy rules (Criterion E). Inter-rater reliability for diagnosis was .78. The clinical reappraisal subsample was weighted to be representative of the U.S. population in the age range of the sample (18–44 years). A detailed description of the clinical reappraisal procedures is reported elsewhere (1, 19). We grouped the ADHD diagnoses into three mutually exclusive categories: never met diagnostic criteria, met full childhood criteria with no current symptoms (childhood ADHD), and met full childhood criteria with current symptoms (adult ADHD).

Major depressive disorder was diagnosed using the CIDI. Diagnoses were based on DSM-IV criteria. Two non-overlapping probability samples were selected, one to complete a clinical reappraisal interview to evaluate lifetime diagnoses, and a second to complete a clinical reappraisal interview to evaluate 12-month diagnoses. The Structured Clinical Interview for DSM-IV (25) was used for clinical reappraisal. Respondents were classified using three mutually exclusive categories: lifetime (meeting criteria for a major depressive disorder at some point during their life), past 12 months (meeting criteria at some point in the past year), or never having met criteria.

The CIDI was also used to assign diagnosis of BED; however, while DSM-IV requires binges to persist for 6 months or more, the CIDI required only 3 months or more. In the present study, respondents were classified into three categories: 1) lifetime, meeting criteria for BED at some point during their life; 2) past 12 months, meeting criteria at some point in the past year; or 2) never having met criteria.

Respondents were asked to bring in medication bottles for all medications and medication status was recorded. Because use of stimulants and antidepressants could potentially influence the results, the presence/absence of each medication type was dummy coded and entered as covariates in the analyses. Antidepressants were coded in two ways: those with a weight gain side effect and all others. Demographic factors assessed included age, gender, race/ethnicity and education level, and smoking status.

Statistical Analysis

Distributions of BMI categories according to major depressive disorder, BED, and ADHD status were compared using Chi-square statistics. Multinomial logistic regression was used to determine the odds of overweight and obesity compared to normal BMI among persons with childhood ADHD and adult ADHD compared to those with no ADHD. A crude model was computed followed by a multivariate model adjusting for age, gender, race/ethnicity, education and smoking status. To explore the possible impact of major depressive disorder and BED on the association between ADHD and BMI, mediation models were conducted via methods developed by Baron and Kenny (26). Accordingly, mediation may be present if the following four criteria are met: 1) the independent variable (ADHD) is associated with the outcome (BMI), 2) the independent variable is associated with the hypothesized mediator (major depressive disorder and BED), 3) the mediator is associated with the outcome, controlling for the independent variable and 4) the association between the independent variable and the outcome variable becomes attenuated or not statistically different from zero when controlling for the mediator. Mediation models were derived separately for BED and major depressive disorder. Each model controlled for demographic factors and smoking status. Although originally proposed for longitudinal designs,

mediational methods (26) may also be valid for cross-sectional data, although inferences of causality are limited (27).

The CPES dataset included respondent sampling probability weights to allow inference to the general U.S. population of 18–44 years. The weights took into consideration the multi-stage sampling methods used in each study and the over-sampling of Part I NCS-R respondents for Part II of the questionnaire. Weights for individual datasets and combinations of datasets were included. The analyses use weights for the combined NCS-R Part II and NSAL dataset.

RESULTS

Sample description

The mean age of the sample was 31 years old (SE=0.25), with 51.6% female (Table 1). The majority (63.9%) were non-Hispanic White, with 16.1% Hispanic, 12.7% non-Hispanic Black and 7.4% Asian or other race, reflecting the population weighting of the data. Greater than half (54.1%) had attained education beyond high school. Nearly one-third (29.2%) were smokers.

Prevalence of DSM-IV diagnoses

Prevalence of childhood and adult ADHD was 7.3% and 3.6%, respectively. Among individuals with ADHD in childhood only, 47.3% were inattentive type, 26.0% were hyperactive type and 26.7% were combined type. Among individuals with adult ADHD, 35.4% were inattentive type, 26.3% were hyperactive type and 38.3% were combined type. Major depressive disorder past year prevalence was 8.4% with lifetime prevalence of 17.9%. BED prevalence past year prevalence was 2.0% with lifetime prevalence of 4.9%.

BMI distribution

Almost half (48.9%) had BMI \leq 24.9 (underweight/normal), 29.2% had BMI between 25.0 and 29.9 (overweight), and 21.9% had BMI \geq 30.0 (obese) (Table 2). Obesity was more prevalent among persons with adult ADHD (29.4%) than among those with a history of childhood ADHD but no adult symptoms (23.7%) and those with no history of ADHD (21.6%) ($P < .006$). Prevalence of obesity (BMI \geq 30.0) was higher among persons with a diagnosis of BED in the past 12 months (44.6%) than among those with lifetime diagnosis (26.4%) and those with no history of BED (21.7%) ($P = .02$).

Multinomial logistic regression models

In the crude multinomial logistic regression (model 1), adult ADHD was associated with statistically significant increases in the odds of being overweight (odds ratio (OR)=1.58; 95% confidence interval (CI)=1.05, 2.38) and obese (OR=1.81; 95% CI=1.24, 2.64) (Table 3). These associations were similar in model 2, which adjusted for demographics and smoking (overweight OR=1.61; 95% CI=1.01, 2.57; obese OR=1.81; 95% CI=1.19, 2.57). Childhood ADHD was not associated with overweight or obesity.

Mediation models

The criteria for step 1 of the mediation analyses for both past year major depressive disorder and BED were met, since adult ADHD was positively associated with both overweight and obesity. Lifetime variables (as opposed to past year) were not associated, and were thus not tested for mediation. Adult ADHD was significantly associated with past year major depressive disorder (OR=2.97; 95% CI=1.56, 5.65) (criteria 2, see Table 4). Criteria for step 3 was also met, major depressive disorder in the past year was independently associated with

obesity (OR=1.38, 95% CI=1.02, 1.85). Step 4 criteria was not met. Adult ADHD remained associated with overweight (OR=1.57; 95% CI=0.99, 2.70) and obesity (OR=1.69; 95% CI=1.01, 2.82) when controlling for major depressive disorder.

Adult ADHD was significantly associated with past year BED (OR=5.1; 95% CI=2.83, 10.72) (step 2, see Table 4). Adults with ADHD had rates of past year BED (10.2%) that were six-fold that of adults with no history of ADHD (1.7%). Controlling for ADHD, past year BED was associated with obesity (OR=2.89, 95% CI=1.21, 6.92), but not with overweight (OR=1.54, 95% CI=0.62, 3.82). The association between adult ADHD and obesity was no longer statistically significant when controlling for BED in the past 12 months (1.41, 95% CI=0.76, 2.53).

DISCUSSION

This is the first population-based study to examine the association between ADHD and obesity in adults. Results revealed that having adult ADHD increased the odds of being overweight and obese. Among individuals with adult ADHD, 33.9% were overweight and 29.4% were obese, compared to 28.8% and 21.6% respectively, of individuals with no history of ADHD. Past year or lifetime history of major depression did not affect these associations; however, past year BED mediated the associations between ADHD and both overweight and obesity.

Findings might suggest mechanistic overlap between ADHD and obesity, which has been alluded to elsewhere (28, 29). One possible neurobiological mechanism is that both ADHD and obesity are linked to hypo-dopaminergic function in the brain (30). In ADHD, hypo-dopaminergic function influences the prefrontal cortex, which is important for sustaining and dividing attention as well as inhibiting distraction (31). Dopamine is also central to appetite regulation. Risk taking, substance abuse, and eating pathology have been associated with DRD2 receptor dysfunction (32). Both ADHD and obesity have been characterized by deficiencies in the DRD2 receptors (28, 32). Davis and colleagues (29) have suggested that because palatable energy-dense foods are known to activate dopamine pathways, overeating among individuals with ADHD may be an attempt at self-medication. Self-medicating behavior in ADHD including alcohol and other substance abuse has been well-documented (33–35).

ADHD symptoms might have implications for weight regulation. ADHD has been characterized as a problem of behavioral disinhibition affecting memory, motivation, and self-regulation of affect (36). As a result, an individual with ADHD might fail to plan meals ahead of time, frequently skip meals due to distractions, and/or lose sight of intentions to moderate food intake. Difficulties with sustaining complex behaviors have been seen in persons with ADHD and are most notable when there is no immediate gratification involved (37). Weight regulation is exceedingly difficult, requiring a great deal of behavioral inhibition in light of the current obesogenic environment (29), and would seem especially challenging for individuals with ADHD.

A diagnosis of BED in the previous year mediated the relationship between adult ADHD and obesity. Adults with ADHD have difficulty regulating their emotions, and such difficulties may lead to eating reactively in response to unpleasant moods. Findings are also consistent with a study that revealed that ADHD symptoms were associated with eating in response to negative moods and environmental cues, as well as binge eating (29). Dysregulated eating could be a potential pathway between ADHD and obesity. The present study is the first population-based study revealing a comorbidity between ADHD and BED.

The present study has several limitations. Height and weight were assessed via self-report, leading to underreporting due to social desirability biases (38). Features of ADHD such as impulsivity and distractibility might result in less frequent self-weighing and therefore less accurate self-reports in those with ADHD. Infrequent self-weighing could lead to underestimated self-reports since weight tends to increase over time. Both potential sources of misclassification would bias the association between ADHD and obesity toward the null. Also, BMI was only available as a categorical variable in the CPES dataset. There are limitations to the diagnostic measure of adult ADHD as discussed elsewhere (1) including reliance on DSM-IV criteria for ADHD which were developed for children, and that direct evaluation was performed on a reappraisal subsample with imputations performed for the remaining cases. However, no standard clinical evaluation of adult ADHD currently exists. Another limitation is that although we test the mediating effect of depression and BED, causality cannot be established in cross-sectional studies. Despite the large total sample size, the prevalence of conditions under study was low making it difficult to assess interactions of major depressive disorder and BED on the association between ADHD and BMI or the influence of ADHD subtypes on risk for obesity.

Results suggest that ADHD is linked to both obesity and BED in the US adult population. The presence of ADHD in overweight and obese patients has clinical implications, including difficulty with behavioral weight management skills such as self-monitoring, meal planning, and adhering to nutrition and physical activity goals. Given that BED mediated the association between ADHD and obesity, dysregulated eating patterns among adults with obesity and ADHD should be studied further.

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Table 1

Description of study sample (n=6,737)*

| | Mean (SE) |
|--|------------|
| Age (years) | 31.0 (.25) |
| | % |
| Gender (female) | 51.6% |
| Race/ethnicity | |
| Asian/other | 7.4% |
| Hispanic | 16.1% |
| Non-Hispanic Black | 12.7% |
| Non-Hispanic White | 63.9% |
| Education | |
| Less than high school | 14.2% |
| High school degree | 31.7% |
| Post high school degree | 54.1% |
| Current smoker | 29.2% |
| Stimulant medication | 0.61% |
| Anti-depressant/no weight gain | 2.9% |
| Antidepressant/weight gain | 5.5% |
| Major depressive disorder | |
| Never | 82.1% |
| Lifetime | 9.5% |
| Past 12 months | 8.4% |
| Binge Eating Disorder (n=5,215) | |
| Never | 95.0% |
| Lifetime | 2.9% |
| Past 12 months | 2.0% |
| ADHD** | |
| Never | 92.8% |
| Childhood only | 3.7% |
| Adult (requires a childhood diagnosis) | 3.6% |

* Data weighted to account for complex sampling frame and to represent national U.S. adult population.

** These are mutually exclusive categories. A total of 7.3% met criteria in childhood, regardless of adult status.

Table 2

Distribution of BMI by categories of ADHD, major depressive disorder and BED.*

| | Body mass index | | | P-value |
|--|--|---|---|---------|
| | Normal (≤ 24.9 kg/m ²) | Overweight (25.0–30.0 kg/m ²) | Obese (≥ 30.0 kg/m ²) | |
| Total sample | 48.9% | 29.2% | 21.9% | - |
| ADHD | | | | 0.006 |
| Never | 49.6% | 28.8% | 21.6% | |
| Childhood only | 42.4% | 33.9% | 23.7% | |
| Adult (requires a childhood diagnosis) | 36.8% | 33.9% | 29.4% | |
| Major Depressive Disorder | | | | 0.25 |
| Never | 48.8% | 29.8% | 21.4% | |
| Lifetime | 46.6% | 28.8% | 24.6% | |
| Past 12 months | 49.3% | 25.8% | 24.9% | |
| BED (n=5,215) | | | | 0.002 |
| Never | 48.8% | 30.0% | 21.7% | |
| Lifetime | 38.4% | 35.2% | 26.4% | |
| Past 12 months | 31.0% | 24.5% | 44.6% | |

Table 3

Multinomial logistic regression models of the association between attention deficit hyperactivity disorder and overweight and obesity.*

| | Overweight (vs. normal) | Obese (vs. normal) |
|-----------------------------------|--------------------------------|----------------------------|
| | Odds Ratio (95% CI) | Odds Ratio (95% CI) |
| <i>Unadjusted Model</i> | | |
| ADHD | | |
| Never | 1.0 | 1.0 |
| Childhood | 1.38 (0.88, 2.15) | 1.27 (0.76, 2.11) |
| Adult | 1.58 (1.05, 2.38) | 1.81 (1.24, 2.64) |
| <i>Adjusted Model⁺</i> | | |
| ADHD | | |
| Never | 1.0 | 1.0 |
| Childhood | 1.19 (0.75, 1.90) | 1.20 (0.71, 2.03) |
| Adult | 1.61 (1.01, 2.57) | 1.81 (1.19, 2.76) |

* Data weighted to account for complex sampling frame and to represent U.S. adult population.

⁺ Adjusted for demographics and smoking status.

Table 4

Multinomial logistic regression models of the association between ADHD and overweight and obesity, adjusting for major depressive disorder and BED*

| | Overweight (vs. normal) | Obese (vs. normal) |
|---|--------------------------------|---------------------------|
| <i>Depression Adjusted Model</i>⁺ | | |
| | Odds Ratio (95% CI) | Odds Ratio (95% CI) |
| ADHD | | |
| Never | 1.0 | 1.0 |
| Childhood | 1.17 (0.75, 1.84) | 1.14 (0.68, 1.89) |
| Adult | 1.58 (1.00, 2.48) | 1.70 (1.12, 2.56) |
| Major Depressive Disorder | | |
| Never | 1.0 | 1.0 |
| Lifetime | 1.04 (0.74, 1.47) | 1.23 (0.95, 1.59) |
| Past 12 months | 1.06 (0.82, 1.36) | 1.30 (1.02, 1.67) |
| <i>BED Adjusted Model</i>^{+ #} | | |
| ADHD | | |
| Never | 1.0 | 1.0 |
| Childhood | 0.88 (0.51, 1.51) | 1.05 (0.54, 1.84) |
| Adult | 1.05 (0.54, 2.05) | 1.41 (0.76, 2.52) |
| BED | | |
| Never | 1.0 | 1.0 |
| Lifetime | 1.62 (0.88, 2.96) | 1.43 (0.85, 2.42) |
| Past 12 months | 1.54 (0.62, 3.82) | 2.89 (1.21, 6.92) |

* Data weighted to account for complex sampling frame and to represent U.S. adult population.

⁺ Also adjusted for demographics and smoking status.

[#] n=5,215